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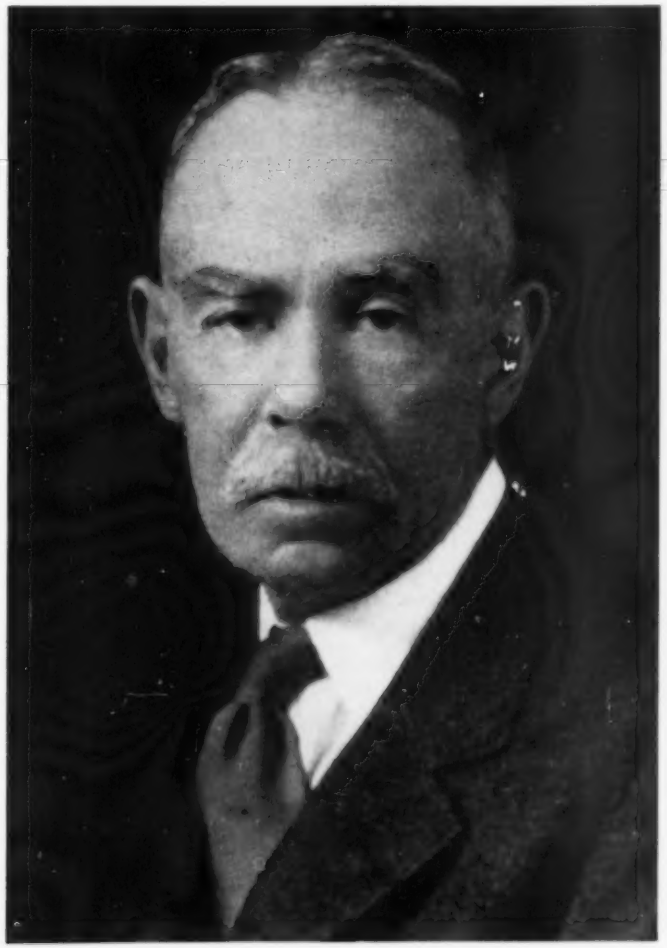
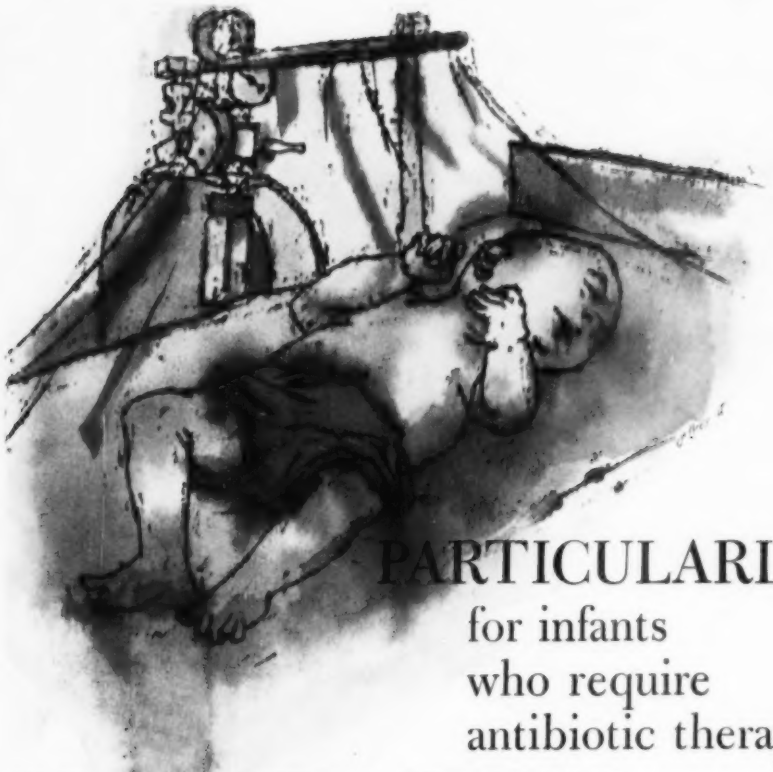


Pioneers of Pediatrics . . . John Lovett Morse (1865-1940)



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Vol. 76, No. 11

November 1959

CONTENTS

The Syndrome of Transposition of the Great Arteries
Elvira Ostlund, M.D. 427

Concern about Radiation in X-Ray Diagnosis—Real or Unreal
Herbert V. von Gal, M.D. 437

Traumatic Multiple Hyperostosis of Bone
C. C. de Silva, M.D. and Susantha Jayaratne, M.D. 447

Neonatal Amegakaryocytic Thrombocytopenia
Harold E. Stadler, M.D., et al 450

Advantages of a New Salicylate in Immunization Reactions in Infants
Emanuel Dubow, M.D. 455

Books 458

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Cover — John Lovett Morse (1865-1940) — see page 13

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1. Phelps, W. M.: Arch. Pediat. 76:243 (June) 1959. 2. Spears, C. E., and Phelps, W. M.: Arch. Pediat. 76:287 (July) 1959.

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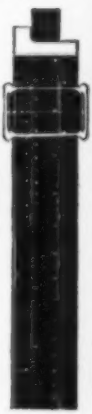

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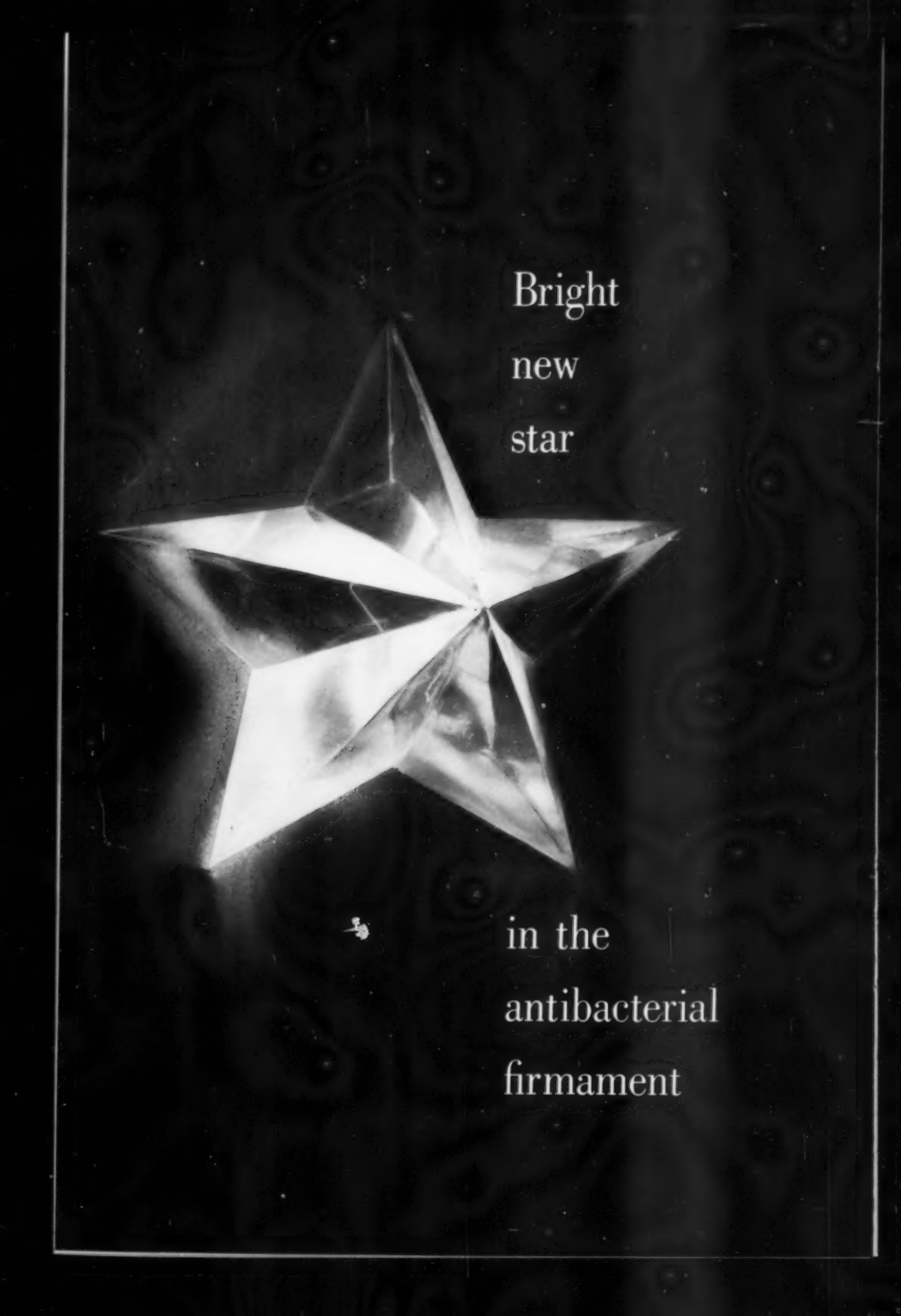
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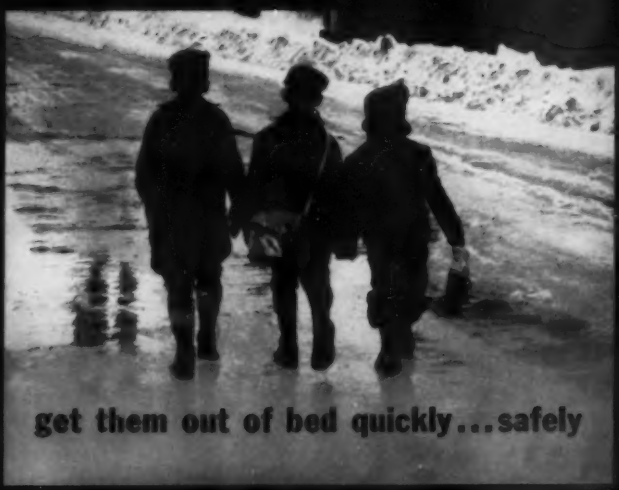

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The Cover . . .

John Lovett Morse (1865-1940)

(Portrait, courtesy of "Journal of Pediatrics")

An important leader in the development of pediatrics in America, Doctor Morse entered the field from general practice. Interesting to note, he was one of the early applicants for certification. Dr. Morse held certificate No. 1 by vote of the American Board of Pediatrics! He was later to have distinction as one of the founders and presidents of the American Academy of Pediatrics, and President of the American Pediatric Society.

Dr. Morse was among the first to limit his work to children. His approach was distinctly clinical, with an acumen and skill that earned him widespread reputation. Much of his time was given to hospital work, teaching and writing.

His teaching career took him from instructor in children's diseases at Harvard, to appointment in 1915 as professor of pediatrics, later becoming professor emeritus. He was chief of the medical staff of the Children's Hospital, and Infant's Hospital, as well as consulting pediatrician to other Boston institutions.

From much of what is written about Dr. Morse, there is one salient point: he will always be marked as an unforgettable figure, liked and respected for the work he did, and held in high esteem for his deserved position in the days when pediatrics, as a special field of medical practice in America, was in the development stage.

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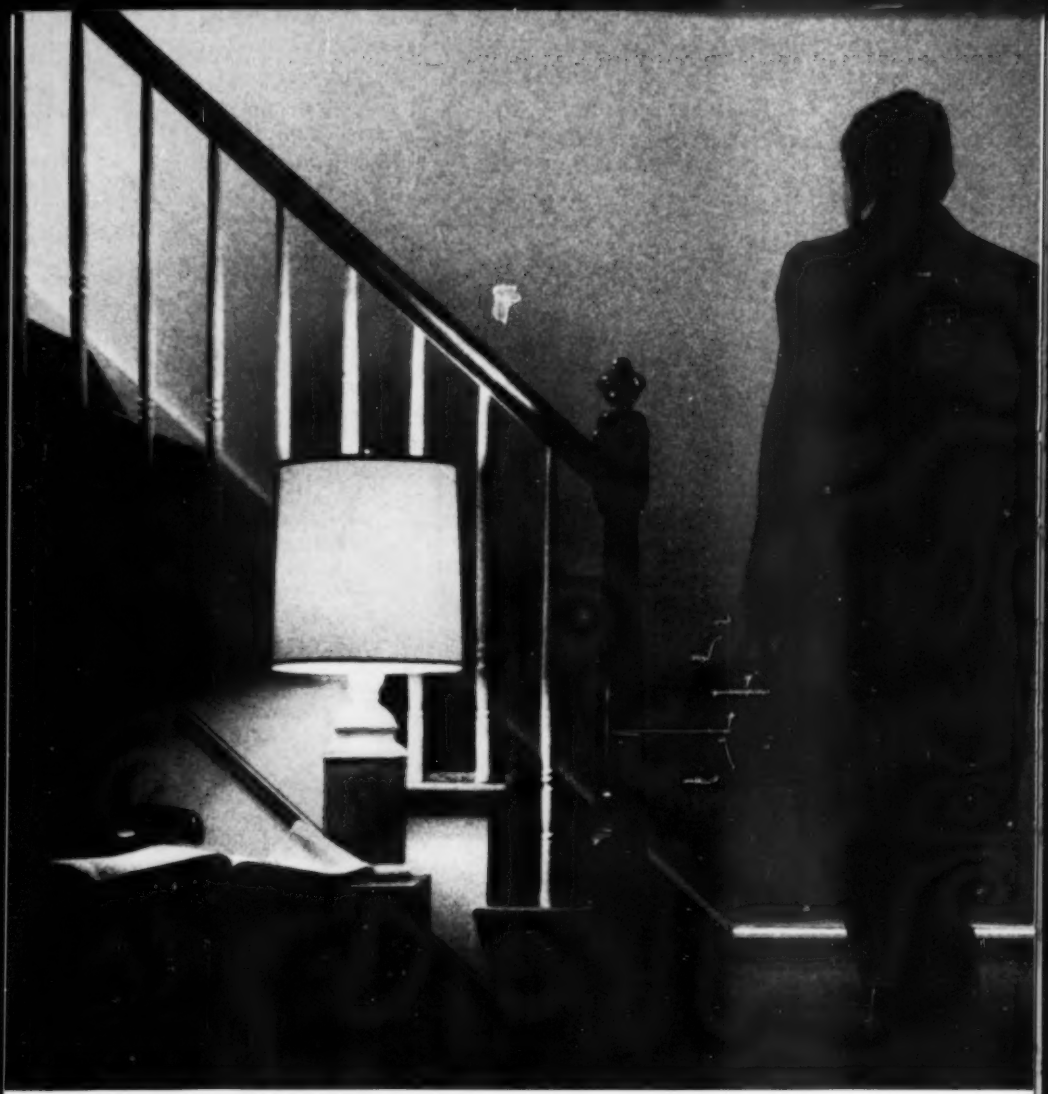

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ARCHIVES of PEDIATRICS

November 1959
Vol. 76 No. 11

THE SYNDROME OF TRANSPOSITION OF THE GREAT ARTERIES

ELVIRA OSTLUND, M.D.*

New York

CASE REPORT

A full-term female infant, R. S., was born at United Hospital to a young, healthy, unmarried, colored woman. No history was obtained of any illness of the mother or of any disorder during early gestation. This was the second pregnancy; the first had resulted in abortion about the third month. Subsequent to this birth the mother has been delivered of another full-term infant, normal except for supernumerary digits. A maternal aunt had a similar defect, but there were no other known familial anomalies.

The infant weighed 7 lbs. at birth and was found normal on physical examination with the exception of a heart murmur. A harsh and loud systolic murmur was heard over the precordium, maximally in the third left intercoastal space. There was no respiratory difficulty, and no cyanosis was visible. Cord Wassermann was negative. Baby regained her birthweight in six days, and therefore was discharged with the mother at that time. As the mother's place of residence was located outside the hospital district, baby, on discharge, was referred through Social Service to the Cardiac Clinic at the county hospital (Grasslands). Subsequently infant was seen at said clinic.

At 12 weeks of age baby was readmitted to United Hospital with acute respiratory symptoms simulating pneumonia. To mother's knowledge baby had thrived and been well till the day prior to admission when nasal discharge and cough developed. On admission baby was promptly treated for the acute infection and it was later determined that she was in congestive failure. She had low-

* Consulting Pediatrician, St. Luke's Hospital, New York City. Director of Pediatric Cardiac Clinic, United Hospital, Port Chester, N. Y.

grade fever, anorexia, cough, restlessness, slight cyanosis, dyspnea with retraction, and inconstant crackling pulmonary rales. In addition there was tachycardia (208 per min.), cardiomegaly, some periorbital edema, a palpable liver, and a gallop rhythm, together with a harsh, grade 3 systolic murmur along lower left sternal border. The child was digitalized and after she had been stabilized on the drug, the heart was found to be enlarged and over-active (140-160 per min.). Tachypnea was present, but there was now no chest retraction, and no visible cyanosis. The harsh systolic murmur was audible over the whole precordium and transmitted to the back; but it was best heard in the third and fourth intercoastal spaces near the sternal edge. P_2 was present and duplicated. By x-ray (fig. I) the heart was generally enlarged with a convex left

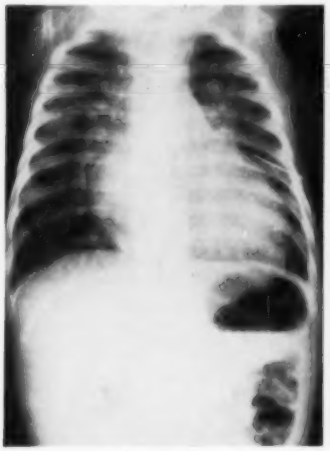

FIG. I—L

FIG. I—R

border, a downward and laterally pointing apex; and there was increased convexity to the right of the sternum, as well as evidence of left atrial enlargement. Lung vascularity was amplified, and there was a striking increase in the hilar vascular markings. The ECG (fig. II) showed right axis deviation, dominance of the right ventricle, and increased height of the peaked P waves in Lead II.

The child's stay in the acute hospital had to be prolonged due to the fact that she developed diarrhea while there. Though very ill, she recovered and was then transferred to St. Luke's Convalescent Hospital, weighing 10 lbs. 6 oz. at 4 months of age or 4 oz. above her admission weight a month earlier. This is 76% of the normal expected weight for age.

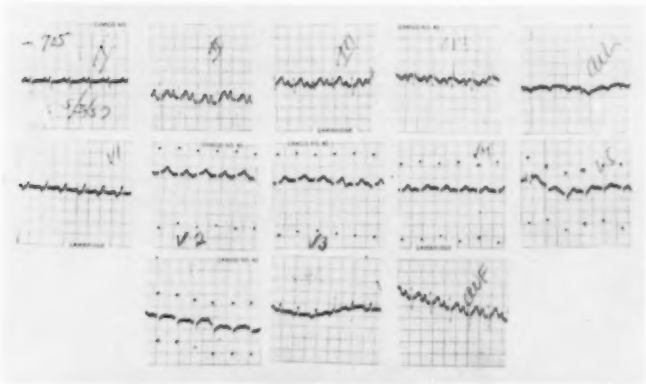

FIG. II

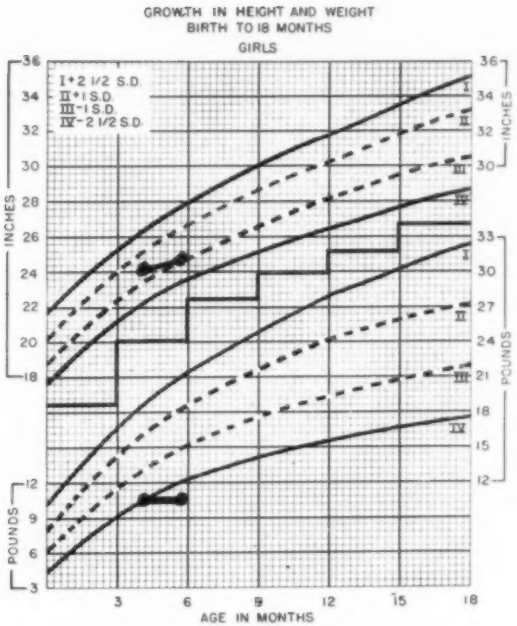

FIG. III

At the Convalescent Hospital digitalis maintenance was continued; and child was kept free of infection. She became more responsive, but her physical development and growth lagged increasingly (Fig. III). At times a diastolic murmur was heard leftward of the sternum. Systolic blood pressure was 90; Hb. 9.6 gm.; R.B.C. 5,500,000. The baby spent considerable time on a terrace adjoining the nursery, and in that favorable lightexposure it was first noticed that she became faintly cyanotic at times, even while at rest. The cyanosis gradually increased in degree and frequency during the subsequent 2 months. It was then suspected that her congenital lesion was something more than a ventricular septal defect which heretofore had been the diagnosis. For that reason she was sent in to St. Luke's Hospital, N. Y. for cardio-respiratory studies. These were done by Dr. A. L. Loomis Bell, Jr. when child was 6 months of age.

On catherization the pulmonary artery could not be entered from the right ventricle. There was high right ventricular pressure. From what appeared to be the region of the superior vena cava catheter passed into a vessel (anomalous) which led into the right lung. Oxygen saturation and pressure values are shown in the following table:

	O_2 Content (cc%)	Pressure (mm Hg.)
Rt. Vent.	7.9	79/4, 89/3
Rt. Atrium	10.7, 8.1	4
S.V.C.	5.1	
I.V.C.	5.9	
Lt. Pulm. Vein or Lt. Atrium	16.25	
Rt. Pulm. Vein	14.25	
Brachial Artery	9.85 -- 48.25	Saturation

Angiocardiogram — in A-P plane only — (Fig. IV) showed prompt and complete filling of the aorta from the right ventricle when but a mere trace of dye showed in the pulmonary artery.

On completion of these studies baby was returned to the Convalescent Hospital where her status remained unchanged for the next 2 months. Then a viral type of respiratory infection (Asian Flu) gained entrance to the institution, and the patient promptly developed the malady.

Again she showed signs of failure. Digitalis was adjusted; she was given oxygen, various antibiotics, and special nursing care.

After a fortnight she improved, but the improvement was of short duration. In a week she relapsed. There was increase in cyanosis and polycythemia (Hb. 15 gm., R.B.C. 9,000,000), and signs of failure worsened. This time she did not respond to any treatment. She died, 9 months of age.

FIG. IV

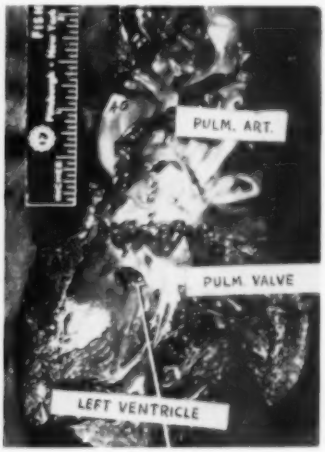

FIG. V

The cardio-pulmonary studies had been considered incomplete inasmuch as the catheter had not entered an efferent vessel from the right ventricle. Despite this, the studies, together with the clinical facts, supplied the necessary information. Briefly stated these features were: Infant with significant murmur from birth thrived in the postnatal period; but at 4 months an acute respiratory infection precipitated failure, and heart was then enlarged. Central cyanosis appeared shortly thereafter and increased. Growth diminished. Partial pulmonary drainage to right heart was demonstrated, but there was no left-to-right shunt found below the atrial level. Right ventricle had high pressure. Aorta filled completely from this chamber but pulmonary artery showed no direct supply therefrom.

The postmortem examination was done in the pathology department at St. Luke's Hospital, N. Y. by Dr. Nora Spens. Photograph of the heart was provided through the kindness of Dr. Rudolf Garret of that department. The findings were as follows:

Heart weight: 93 gm. (Normal 35 gm.)

One vein (cut off with lung) led into the auricle of the right atrium. Vessel was presumed to originate from the right lung as

it was located in association with right branch of pulmonary artery. Left common carotid arose from right innominate. Left subclavian arose from the aorta, but it was a vessel of unusually small caliber. Pulmonary artery originated immediately to the left of the aorta, and for a distance of one inch these two vessels lay back-to-back, densely adherent. The Ductus was identified as a fibrous cord between the two.

Both atria were dilated. Foramen Ovale was probe-patent but functionally closed. A defect of 7x3 mm. was present high in the muscular part of the interventricular septum at the muscular membranous junction. Rt. ventricle was markedly hyperthrophied (each ventricle 1 cm. in thickness). The aorta with coronary ostia and normal valve leaflets arose from the right ventricle and curved to the left in the normal way. The pulmonary artery came off from the left ventricle, and its valve cusps were thickened with some calcification of commisures (Fig. V).

Distribution of the coronary arteries was normal; but the origin of the aorta was rotated, so that the left coronary artery arose from the cusp which was posterior and the right coronary arose from the left anterior cusp. Both coronary ostia were larger than normal.

Lungs were congested, and both lower lobes were solid. The only vestige of an inflammatory process was seen in the smaller bronchi which showed desquamation and inflammatory exudate.

Other organs were normal.

DISCUSSION

Complete Transposition of the Great Arteries of which this case is an example is recognized as a relatively common cause of failure and death during the early months of life in cyanotic infants with cardiomegaly.

Embryologically the defect is an early one, and it is centered in the core of the heart. When, in the course of development, a septum forms in the primitive tubal heart and the single bulbar trunk that leads off therefrom, it is necessary for that septum to form in such a way that the two circulations will cross. If this is not contrived, venous blood will not enter the pulmonary circuit nor oxygenated blood the systemic; instead there will be two "parallel" independent circuits. This crossing of the circulations is accomplished in the bulbus cordis by specific torsion of that chamber[6,7,8,10]. Such rotation at the efferent end of the heart spirals the bulbar septum which divides the two arterial trunks and this permits normal connection so that

the vessel from the right ventricle leads off to the lung and the vessel from the left ventricle becomes the systemic trunk. If this specific torsion fails, the normal absorption of the primitive bulbus is interfered with and we have this arrest called Transposition. In Transposition there may be various degrees of displacement of the origin of the two big vessels, but the most common is Complete Transposition as in this case with aorta arising from the right ventricle and pulmonary artery from the left.

Because of this abnormal take-off of the two arterial trunks from the heart Complete Transposition is a grave lesion. In the first place, the systemic circulation is supplied by venous blood which recirculates from the right ventricle. Our case had a scant 50% oxygen saturation. This is inadequate for growth as our patient demonstrated. Secondly, the coronaries, too, get venous blood from the aorta; and, beyond doubt, this is a major factor which brings on the early failure, so frequently a feature of this anomaly. Then, too, in cases with but one opening between the two circulations (such as ventricular septal defect), and especially if the defect is not a large one, then there is a third source of strain on the heart due to the meagre and difficult shunt. In these cases oxygenated blood can be shunted to the aortic side only when the right ventricle has pumped enough blood through the defect into the pulmonary side to raise that pressure above the systemic. It is obvious that such a scanty supply of aerated blood, obtained in this devious way, constitutes a precarious lifeline to the patient and, in turn, demands a tremendously increased cardiac output.

Our case was actually a Complete Transposition with Complications in view of the added vessel anomalies. The anomalous pulmonary vein, a favorable defect in this case, which brought oxygenated blood to the right heart, gave our patient a fair oxygen saturation and it explained the lack of early and deep cyanosis so commonly present. This steady inflow of saturated blood from one pulmonary vein must of necessity have caused the ventricular shunt to be a right-to-left shunt (fitting the right ventricular saturation figure).

To a certain extent, duration of life in these transposition cases is dependent on the intra- and extra-cardiac communications. Our case had two such communications, a ventricular septal defect of fair size and one pulmonary vein emptying into the right heart, neither of which is a "closing" type of defect. From this standpoint our patient was better off than most. The set-up of this transposi-

tion seemed compatible with a longer life. Yet her course was not too different from that of the average transposition.

To be sure, she survived two infections, one respiratory and one enteric. For each she was given specific antibiotic therapy. For the final infection no specific treatment was available (nor was the influenza vaccine available prior to the time she developed the illness). For this viral respiratory infection the patient was a bad risk. In this case then, the external factor, Asian Influenza, influenced the course.

Most transpositions die early in infancy. 85% of Keith's series[17] from the Hospital for Sick Children at Toronto had died during the first 6 months of life; and Nadas' series[18] from Boston Children's Medical Center showed that only 8% survived by the end of the first year. This high mortality and rapid course has stimulated various surgical attempts at treatment. In 1950 Blalock and Hanlon[12] published their operative results on 33 transpositions. They used extracardiac shunts on the majority, but in some they created an atrial septal defect. In others they used a combination of the two, and they felt that the combination gave the best results. The authors did not consider their results striking, however. But it is interesting to note a recent report from Children's Hospital, Philadelphia, by Ash et al[21]. In their transpositions they report better results in the cases which were given an artificial atrial septal defect than in the cases treated by other surgical methods. In 1955 Lillehei et al[14] published their series of 32 cases which they had operated on using venous transplant procedures (pulmonary vein to right auricle, and in some cases the additional shunting of cava to left auricle). At the time of publication of that study only 22% had survived the operation more than a year.

Baffes[20] has pursued the method of transposing the venous return; and he has planned his procedure as a two-stage operation. In the first stage the inferior cava is anastomosed to the left atrium, and the right pulmonary veins to the right atrium. This gives the patient better than a 50% correction in oxygenated blood supply as it crosses that much of the circulation. Second stage is planned to complete venous transposition. The mortality is over 30% for the first-stage procedure. Other methods have been tried[18,22].

If one regards carefully the hearts of transposition cases that have been thoroughly studied[5, 7, 9, 10], one finds more malformation than the name implies. Misplaced or transposed origin of the two

arterial trunks is not the only derangement. For survival there must be at least one communicating defect; but there are other evidences of disorganized structure. There may be, and there usually are abnormalities at the cardiac ostia. The semilunar valves are often involved. Commonly there is pulmonary valve obstruction as in our case. At times the obstruction is located in the right ventricular outflow area, e.g. in the infundibulum or at the crista supraventricularis. The atrioventricular valves are sometimes inverted. Moreover, the ventricles are at times disorganized; septation may be incomplete or there may be false septation. Sometimes the muscle bundles of the right ventricle are abnormal. Coronary vessels may be inverted or malpositioned.

Altogether this defect, due to faulty absorption of the bulbus cordis[1,4,6,7,9,10], is a comprehensive defect with a variety of manifestations. Sir Arthur Keith aptly labeled it a "syndrome".

It is easy to understand that a heart so fundamentally handicapped should fail early. It is also obvious that surgical treatment at best can only ameliorate this type of anomalous heart. It is an inefficient organ full of handicaps and cannot become the smoothly propulsive organ that will function as the normal heart.

Therefore, prevention must be the ultimate aim. But prevention is not possible as long as we are ignorant of what to prevent. As yet we do not know if this defect can be acquired; and if so, whether some factor in the mother or the mother's environment can be the cause. If there is a maternal environmental cause, then practicing physicians are the ones who can ferret out this and establish the relationship.

But it is not the pediatrician nor the cardiologist attending the infant with transposition who can get this information. By the time the infant's heart defect is discovered and diagnosed, the circumstances of the mother at that time in pregnancy when the defect occurred, will have been forgotten. It is rather the physician attending the mother during pregnancy who can with some accuracy obtain such detailed information on her environment. To be of value this information must be minute and meticulous and it must be obtained at the first pregnancy visit.

As a rule, the practitioners who engage in obstetrics are primarily interested in the climactic delivery and in that part of the pregnancy which affects the mother. It is difficult to stimulate their interest in that intangible period of pregnancy, the first tri-

mester which is more significant to the offspring than to the mother. And it is difficult to stimulate their interest to such a point that they will in each case give painstaking time to a questionnaire which will review the mother's total environment. Yet, this would be a direct way to the answer.

Group pediatricians practicing with physicians who give prenatal care are ideally situated to stimulate such interest in that important but unfocused gestational period, the third to eighth fetal weeks, when the structure of the heart, as well as of the other organs, is formed.

It is hoped that this case report will influence some group pediatrician to start such a search.

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64 *Highland Road, Rye, N. Y.*

CONCERN ABOUT RADIATION IN X-RAY DIAGNOSIS . . . REAL OR UNREAL

HERBERT V. VON GAL, M.D.*

Connecticut

It is often difficult in this day and age not only for the public to decide what is actually to be considered dangerous in relation to radiation, but also for the busy physician to evaluate all the technical information and to apply it in relation to his own practice. Understanding a "rad" is of little help in deciding whether an amount of radiation is dangerous either to his patient or himself. The confusion is multiplied by the lack of definite figures that can be given as to the maximum dosage that a person may receive without danger. In this brief paper, I hope to explain with a minimum of detailed radiation physics the relationship of radiation exposure in diagnostic X-rays to the pediatrician and his patient.

Many patients and physicians who read newspaper articles on the occurrence of leukemia, cancer or even sterility of patients following X-ray exposure fail to differentiate diagnostic X-rays from the therapeutic. Although radiation in large doses can unquestionably cause such somatic changes as erythema, loss of hair, sterility, death of epiphyseal centers in a growing child, depression of bone marrow, leukemia, cancer, and death of the individual, none of these somatic changes should ever be of any real concern in properly used diagnostic X-rays.

I know of no verified reported cases of sterility, carcinoma, leukemia or other lymphomas that can be definitely attributed to normal diagnostic X-ray procedures. While therapeutic terminology usually deals with hundreds or thousands of roentgens, the diagnostic procedures usually deal with small fractions of a single roentgen. If then, normal diagnostic X-rays are not a major concern in producing malignancy or sterility, the major consideration is the possibility of gonadal exposure and genetic change and, therefore, of immediate interest to the pediatrician. Whereas somatic changes are limited to the individual and stop with the death of the individual, genetic changes may have lasting effect and pass on from generation to generation.

*Associate Radiologist, Greenwich Hospital, Greenwich, Conn.

When considering X-ray dosages to a person, we must realize that exposure to radiation type energy is not new but man has been exposed continuously to it since the world began. This type of radiation is called natural background. This consists of cosmic rays plus the radioactive materials in our body and the environmental exposure from natural occurring radioactive substances in the earth. Added to this naturally occurring continuous radiation exposure, is the man-made exposure of fall-out from atomic weapon testing, medical and dental X-rays, television, radioactive luminous dials, certain glassware, eyeglasses, etc.

TERMINOLOGY

It is necessary to explain briefly the terminology used in relationship of one term to another. It is simplest to consider a roentgen (r) as a specific quantity or amount of radiation.* One milliroentgen (mr) is one one-thousandth (1/1000th) of a roentgen. A rad is used to signify the absorption of radiation energy in human tissue. One rad may be considered interchangeable with 1 roentgen and so also their fractions, i.e., milliroentgen or millirad.

Primary radiation from an X-ray machine is that coming directly from the target of the X-ray tube. That portion of the primary beam that is directed out of the tube housing and limited by cones, diaphragms or other collimators is called "the useful beam." The useful beam is that which is used to penetrate the portion of the body to be examined and expose the film. When X-rays strike an object such as body tissue some of the radiation is scattered in all directions similar to the dispersion of a beam of light by fog. This is called scattered radiation and explains why even when the useful beam is confined to the chest of an individual small amounts of scattered radiation reach the gonads.

RECOMMENDED LIMITATIONS OF RADIATION EXPOSURES

The effects of radiation exposure have been of great concern to the radiologist almost since the discovery of X-rays in 1895. The National Committee on Radiation Protection was formed in 1929 upon the recommendation of the International Commission on Radiological Protection and since that time has utilized some of the foremost men in the various fields of medicine, physics, biology

*Roentgen is that quantity of X or gamma radiation such that the associated corpuscular emission per 0.001293 gm of air produces, in air, ions carrying 1 ESU of quantity of electricity of either sign.

and genetics to set forth standards of radiation protection. It was not until 1956, however, when reports by the National Academy of Sciences in this country and the British Medical Research Council in England[1] were made that the general public became alarmed about radiation exposure.

It has been recommended by the Committee on Genetic Effects of Atomic Radiation of the National Academy of Sciences[2] that from conception to thirty years of age the general populations' reproductive cells should not be exposed to an average total accumulative dose of more than approximately 14 roentgens. Of this 14 r total, 4.3 r is natural occurring background and the remaining 10 r is man-made radiation. As stated in the National Academy of Sciences report we are now using approximately 3 or 4 roentgens of medical X-ray during this 30 years. This figure is felt by many to be somewhat high and that the general population is being exposed to much less radiation from diagnostic X-rays than stated in the above report. It is the attempt of manufacturers of X-ray equipment and of the radiologists to always keep the exposure to a minimum.

The total of 10 man-made roentgens admittedly is for the population as a whole and the recommendation for the maximum permissible dosage for those exposed to radiation occupationally is naturally higher. It is recommended that no individual, even though occupationally exposed, should receive more than a total accumulated dose of 50 roentgens to the gonads up to 30 years of age and not more than 50 roentgens additionally up to 40 years of age, or a total of 100 r up to age 40.

In the newest recommendations of the National Committee on Radiation Protection[3], the limits for occupationally exposed workers corresponds to an average of 5 r per year after age 18, of whole body exposure and 1.5 per week of limited portions such as the hands. As stated, in the Manual[4] prepared by the American College of Radiology, if these recommendations are followed out little deleterious effect from radiation is expected—although some effect may be shown, such as statistical shortening of life expectancy, currently estimated at about one day per 1.0 r of whole body exposure.

GENETIC CONSIDERATIONS

In 1927 Muller presented his findings on gene mutations following exposure to ionizing radiations of fruit flies. Although it is

generally agreed that all radiation gene mutations are harmful, it it very difficult to evaluate how harmful. Most all figures of abnormal gene mutations have been obtained from experiences with fruit flies, mice and other lower animals. Data like this is difficult to obtain in humans. Genetic injury from radiation has not, as yet, been conclusively demonstrated in man. It is possible, although not known, that man may have a more stable gene than lower animals. However, as in all the protective measures set up concerning radiation, the most conservative estimates of exposure and limitations are the ones set forth to insure safety.

All radiation induced mutations are cumulative and genetic harm is proportional to the total dose over the reproductive years. Mutations of this type are all felt to be undesirable. In the report of the National Academy of Science[2] it was concluded that the most logical estimate of what would be considered the doubling dose of gene mutations by radiation exposure lies in the range of 30 r to 80 r. If it is considered that 2 percent of all live births in the United States have defects of genetic origin, then if a doubling dose of radiation were given, this would rise and eventually be doubled. This estimate would be based on the premise that every person in the reproductive years would have to be exposed to this amount of radiation and not just a small segment of the population. This, of course, would not take place in one generation but would be spread over many generations. Geneticists have estimated that about half the damage would occur in 30 to 50 generations and approximately ten percent of this would be in the first generation.

Although we should be well aware of radiation induced mutations, we must realize that harmful mutations occur from many sources. These so-called spontaneous mutations have always been with us and may very possibly be greatly increased by such new things in our present life as drugs, smoke, food coloring, insecticides, smog, automobile exhaust, preservatives, etc.[3] Dr. Lars Ehrenberg and his Stockholm University research team have recently reported on experimental work on temperature elevation of the male reproductive organs as the result of wearing clothing and its relation to gene mutation. Thermocouple observations of the scrotal skin in humans both clothed and nude showed variations of 3° centigrade in temperature. Correlating these observations with fruit fly determinations of gene mutations in relation to temperature change, they have made the rather amazing statement that the wearing of clothing in the human may be a hundred to a

thousand times more dangerous from a genetic standpoint than those now estimated for different sources of radiation including atomic testing and necessary medical radiation.

AMOUNT OF GONADAL EXPOSURE IN VARIOUS DIAGNOSTIC X-RAYS

In considering radiation exposures to the gonads of infants and children during diagnostic procedures, it is even more difficult to calculate definite quantities of radiation per individual exposure than in adults. Gonadal exposure in a growing child varies with the size of the patient at the time of exposure. Infants require less radiation for comparable examinations than do older children or adults. It is suggested by Standford & Vance[6] that when the reproductive organs of a child are in the direct beam, the average radiation received is 0.1 to 0.2 of that for adult males, where as for females it might be half the adult quantity. Because of the small size of the patient, it is often difficult to confine the exposure to the area desired in an infant—and only too frequently no attempt is made at all to limit the area of exposure. It is easy to understand whereas the area of exposure on a chest film of an adult would only give small amounts of scattered radiation to the gonads that if the same size area of film were exposed with an infant on the film, the entire body would be exposed and the gonads would be in the primary beam. It is not only the amount of exposure that is important but what area is being exposed. If the same factors are used to expose a skull as a hip, the exposure of the gonads from the former would be negligible whereas the latter may be significant. The majority of diagnostic X-ray examinations of children relate to the head, chest or extremities and less commonly to those areas which have a greater tendency to include the gonads in the primary beam—such as pelvis, abdomen, lower spine and hips. A diagnostic X-ray examination of a child's skull, foot, forearm, upper arm or hand give exposures to the gonads of less than 1 mr per examination.[7] This amount of exposure to the gonads is negligible and is approximately that which we may receive every day from natural background radiation.

A single radiograph of the chest taken with proper technique will expose the gonads to approximately 0.3 mr.[4] This is obviously an infinitesimal amount and should be of no concern from a genetic standpoint.

Stomach and upper gastro-intestinal series including both films and fluoroscopic exposures and using the proper techniques will expose the ovaries and also the testes, when not shielded, to a few hundred milliroentgens in a complete examination.[8] During an examination of the lower intestinal tract the gonads will also receive approximately a few hundred milliroentgens.

Diagnostic X-ray examinations such as intravenous pyelograms, lumbar spines, pelvis, and hips may give a total gonadal dosage of approximately 1 r and most of these procedures give much less than 1 r. The use of high voltage or heavy filtration can reduce this exposure considerably. Laughlin & Sherman[9] state that gonadal exposures of the female in pelvic examinations may be reduced to as low as 20 mr. Male scrotal shields markedly reduce gonadal exposure during these types of examinations.

Dental examinations done with the proper equipment can give very low exposures not only to the gonads but also to the skin and bone marrow. Skin doses of less than 0.1 r per film and ovarian doses of 0.06 mr. per film have been reported with 0.34 mr for the testes[9]. Here again, male gonadal dosage can almost be completely eliminated by shielding.

Although pelvimetric studies in the pregnant woman are not directly related to pediatric practice, they are indirectly of great concern to the pediatrician. There appears to be some controversy over the possibility of injury to the embryo or fetus following pelvimetric studies during pregnancy. In 1956 Stewart[10] and his associates put forth statistical evidence favoring the possibility that leukemia occurs about twice as frequently in those children whose mothers had X-ray pelvimetry as those who did not. In 1958 a report from Australia[11] would imply that there is no definite evidence that abdominal X-ray exposures of mothers during pregnancy tend to increase leukemia in the children of these pregnancies. It is interesting to note in relation to whole body irradiation of the fetus that no cases of leukemia have been reported during the first decade in Japanese children who were irradiated in utero by the atomic bomb[12]. In 1957 Robinow and Silverman[13] stated that thus far no fetal malformations had been traced to diagnostic X-ray procedures during pregnancy.

The Russells[14] have stated that as low as 25 r in the early stages of gestation have been effective in producing changes in the developing animal embryo. Fortunately all diagnostic procedure would give exposures well below this figure. Because of the known

sensitivity of the developing cell to radiation, it is generally agreed that if possible pelvimetric studies should be done near term. Routine abdominal X-ray examination that can safely be withheld should be done after delivery. No X-ray examination that is felt necessary for the well-being of a mother or unborn child should ever be withheld.

FLUOROSCOPIC EXPOSURES

Fluoroscopy of any portion of the body gives many times the radiation exposure than a comparable film study of the same area. Unfortunately some pediatricians still use the fluoroscope in routine evaluation of infants and children. One of the most difficult studies of radiology is the fluoroscopic evaluation of an infant's chest. A single chest film will not only give a much clearer and more permanent evaluation of the chest with less distortion but the exposure to the patient is infinitesimal as compared to the fluoroscopic examination. The exposure to the pediatrician is completely eliminated by a chest film rather than fluoroscopic evaluation.

One-minute fluoroscopy of the chest in an adult gives approximately the same total radiation exposure to the gonads as a hundred chest films. Fluoroscopy of an infant's chest using large fields and improper techniques with inadequate equipment undoubtedly gives exposure to the gonads comparable to many thousand properly exposed chest films. It is probably safe to say that nearly one hundred percent of fluoroscopes that have not been recently calibrated by an accredited radiation physicist and his recommendations followed out are giving many times the exposure to the patient necessary for satisfactory fluoroscopic visualization.

Fluoroscopy done with the proper equipment and an experienced fluoroscopist is naturally a very small risk for the information obtained when the examination is necessary. The diagnosis and localization of a tracheo-esophageal fistula in the newborn or the diagnosis and reduction of an intussusception in a child or even the confirmation or denial of a duodenal ulcer in a child with upper abdominal pain are some examples of necessary fluoroscopic examination.

All fluoroscopy should only be done for a very limited few pediatric patients. The routine fluoroscopic examination of either infants or children is to be condemned. Necessary fluoroscopy should only be done by an experienced fluoroscopist who has taken time to accomodate his eyes and only using the proper equipment.

EXPLAINING DIAGNOSTIC RADIATION EXPOSURE TO THE PATIENT

It is difficult to explain radiation exposure to the patient in a few words to allay his or her fears as to its possible dangers in diagnostic work. It is not always comforting to an intelligent person to have a simple negative answer to the question of whether an X-ray will be harmful. It seems to me that the most graphic and comprehensible way of explaining amounts of diagnostic radiation exposure is by relative amounts of comparable every-day natural or common man-made exposures. Table "1" is an attempt to make these comparisons readily available and understandable.

APPROXIMATE GONADAL RADIATION COMPARABLE TO DIAGNOSTIC CHEST FILM EXPOSURE*

1	=	1 Day of wearing a luminous dial watch**
1	=	Difference in gonadal exposure of 1 day living in a brick & concrete apartment as compared to 1 day in wooden house.
1	=	Trip across U. S. in air liner.
1	=	Difference in living one day in Colorado Springs as compared to one day in Pittsburgh, Pennsylvania.
1	=	Average natural background exposure to individual each day.

*Skull, foot, forearm, upper arm and hand examinations may be substituted for chest film.

**Estimated from Libby.[15]

As previously stated, diagnostic radiographs of the chest, skull, foot, forearm, upper arm and hand all give gonadal doses below 1 mr per examination. For all practical purposes, the gonadal exposure from any of these examinations may be considered the same and in comparison may be used interchangeably.

Variations in amounts of radiation exposure by changes in altitude are readily available for comparison, the greater the altitude the more cosmic radiation exposure. Another common exposure comparison is that of the radioactive content of the minerals in a brick and concrete building as compared with the neglibible radio-active content of a wooden building.

The areas of greatest exposure are those which include the gonads in the primary beam. Some of these examinations may give exposures to the gonads up to one thousand mr (1 roentgen). Although these figures are much higher than those for the previously

mentioned examinations of chest, skull, etc., there is no need for concern if the examination is done with the necessary precautions and indications. Many thousands of persons have been exposed to much higher dosages of radiation to the gonads without deleterious effects. In various areas of India the background radiation may be as high as 1000 mr to 2500 mr per year. Preliminary reports have indicated that no abnormal degree of adverse mutational changes have occurred in these people who have lived there for many generations.

CONCLUSION

The problem of minimizing radiation exposure as it relates to pediatrics in X-ray diagnosis is not one of discontinuing necessary informative examinations but the judicious use of proper equipment. This should only be done by well trained personnel who are aware of the benefits as well as the inherent dangers.

Routine fluoroscopic evaluation of infants and children should be discontinued. During fluoroscopic evaluation of an infant done with out-dated equipment and large fluoroscopic fields, the gonads may receive an amount of X-ray exposure equal to many thousand properly taken chest films.

All X-ray equipment should be routinely calibrated by a radiation physicist and his recommendations followed out. A practical and useful guide for the use of X-ray diagnostic equipment is the Manual[4] prepared by the American College of Radiology. More detailed information may be obtained from the National Bureau of Standard Handbooks[3].

Despite the greater use of diagnostic X-ray, much less radiation exposure may be received by the patient per examination than formerly because of the great improvement in the equipment used. Even older equipment with minor changes such as added filtration may reduce the exposure to the patient many fold.

The more common examinations such as chest, skull and extremities give negligible amounts of radiation exposure to the gonads. Examinations around the pelvic area which naturally give higher gonadal exposures are much less commonly used in pediatrics. Naturally, as in all X-ray examinations, they should only be done when specifically indicated and then with the proper equipment and precautions. If this is followed there should be no real concern about radiation exposure in any diagnostic X-ray procedure.

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The long term (8 to 10 years) fate of 40 Mantoux positive infants, aged under three years, who were given antituberculosis chemotherapy for six weeks, has been analysed. Results have shown that such treatment appears to reduce the incidence of complications to the disease and its subsequent relapse in later life, at least before adolescence is attained. Childhood tuberculosis has been discussed, and a plea has been made for the treatment of all recently infected children with a clenotherapy regime which is effective not only in preventing complications, but also in preventing subsequent relapse of their disease.

Author's Summary

TRAUMATIC MULTIPLE HYPEROSTOSIS OF BONE

C. C. DE SILVA, M.D.*

SUSANTHA JAYARATNE, M.B., B.S.**

Ceylon

This is a case of a male child 11 months old who was admitted to the Children's Hospital, Colombo on 25.11.57 with a history of swelling of the limbs of 2 months' duration.

The child was the youngest in a family of 5. The first pregnancy ended in an abortion at the third month. The second and third children are normal and are 6 years and 3 years respectively. The 4th child died at 9 months of age of febrile convulsions. This infant was born at full term in a maternity hospital, delivery being normal. There was no history of neonatal asphyxia. He was breast fed for only 8 days. Thereafter the child was fed on full-cream milk and orange juice was given from the 3rd month; about 3-4 oranges a week. The child was given one dram of milk of magnesia every other day from the age of one month, to the time of admission to hospital. He was in good health until he was 6 months old but at that age he developed a diarrhoea which lasted for about 2 weeks.

When he was 9 months old the mother noticed a swelling of the lower end of the left upper arm which gradually increased in size to involve the entire upper arm. The child would not sleep on the affected side. The swelling felt woody, hard and tender to the touch. This swelling subsided in one week and the right upper and lower limbs developed similar swellings which lasted for 3 weeks.

On admission the child was found to have slight swellings at the lower ends of both upper arms which were tender but not warm nor red. The patient was very irritable and his weight on admission was 13 lbs 12 ozs. These swellings subsided in about a week's time. In about 2 weeks' time the child's irritability disappeared. But he developed a bronchopneumonia and died 8 weeks after admission.

The laboratory findings revealed an increased E.S.R., increased alkaline phosphatase activity with normal calcium and phosphorus levels. Serum sodium, potassium, carbon dioxide and chlorides were within normal limits. The W.B.C. and differential

* Professor of Pediatrics, Univ. of Ceylon.
** Research Ass't., Dept. of Pediatrics, Univ. of Ceylon.

were normal. The Mantoux reaction was negative. The Kahn & Cardiolipin reports were also negative.

E.S.R. (Westergreen)	19.12.57	70 mm 1st hour –1
	30.12.57	30 mm 1st hour –1
	19.12.57	*31.1.58*
White Blood Cell Count	7,800/cmm.	10,800/cmm.
Differential count Polymorphs	45%	59%
Lymphocytes	55%	41%
Electrolytes	*12.12.57*	*28.12.57*
Na	153 mEq/lit.	Serum Calcium 10.0 mg%
K	5.4 "	Serum Phosphorus 4.1 mg%
Cl	107.5 "	Alkaline Phosphatase 16.5
CO_2	22.1 "	(King-Armstrong Units)

X-rays: The X-rays revealed hyperostosis of both humeri and of the right tibia. There was no hyperostosis of the ulnae, parietal and frontal bones of the skull, clavicles or mandibles, pelvis or spine. The 5th, 6th, 7th and 8th ribs on the right side and the 9th on the left side showed fractures with callus formation. Metaphyseal irregularities of both humeri and the right tibia were seen. There were no skeletal abnormalities in the brothers of this patient.

FIG. I

Infantine Cortical Hyperostosis

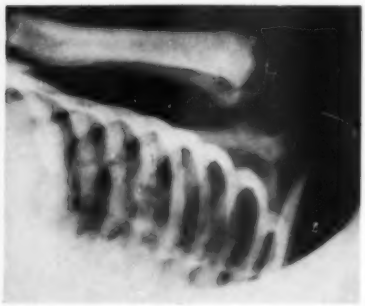

FIG. II

Traumatic Cortical Hyperostosis

Dr. A. W. Atukorale, Radiologist to the Children's Hospital, Colombo suggested the diagnosis of Traumatic Multiple Hyperostosis and this was confirmed by Dr. John Caffey of New York to whom the history, physical and biochemical findings and X-rays were forwarded for his opinion.

POST MORTEM

Findings: Sections of thyroid, parathyroid, testes and liver showed no abnormalities on microscopy. There was a slight increase of fibrous tissue in the pancreas. The lungs showed evidence of bronchopneumonia. The report on the section of bone was that of normal bone tissue with cystic spaces.

DISCUSSION

The child had tender swellings of the lower ends of both upper arms and of the lower end of the right thigh. The swellings gradually subsided. There were no significant laboratory findings. X-rays, however, revealed cortical hyperostosis and terminal metaphyseal irregularities of the humeri and tibia and multiple fractures of the ribs.

Even in the absence of a history of trauma a diagnosis of traumatic hyperostosis of bone was made as cortical hyperostosis and irregularities of the metaphyses are the two most characteristic feaures in this condition. The multiple fractures of the ribs confirm this diagnosis.

Vitamin D poisoning was not considered because there was no history of any added vitamin D having been given to the child, and also the blood calcium and phosphorus levels were normal. There was no evidence of any metastatic calcification.

Infantile cortical hyperostosis (Caffey's Syndrome) was excluded as the metaphysis is never involved in this condition. (Caffey[1].) Trauma is not generally considered as an etiological factor by physicians unless it has been given voluntarily in the history and particularly in the case of children such a history may be suppressed. But actually trauma is a common cause of acute and chronic bone disease. X-rays may be the only means of diagnosing the traumatic origin of the lesions and if the X-ray evidence is sufficiently convincing this diagnosis must be made even in the complete absence of a history of trauma.

Our thanks and acknowledgements are to Dr. A. W. Atukorale, Radiologist, Children's Hospital, Colombo and to Dr. John Caffey of the Babies Hospital, New York for their help in the diagnosis of this case.

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NEONATAL AMEGAKARYOCYTIC THROMBOCYTOPENIA

HAROLD E. STADLER, M.D.*
M. RAY SCHMOYER, M.D.**
JOSEPH P. WORLEY, M.D.*
Indiana

The complex mechanisms involved in the pathogensis of neonatal thrombocytopenia have been greatly clarified in recent years. There is a type of thrombocytopenia associated with hemangiomata[1,2] in which the tumor acts as a filter for the blood platelets, thus reducing the circulating member to pathological level. The question as to whether the platelets are phagocytically removed by the reticuloendothelial system or function in local repair of capillary walls is still unanswered. The presence of blood-forming islands (with megakarocytes) in the tumor walls[2] would speak for a dire local need for platelets.

The second type of thrombocytopenia of the newborn, that resulting from disturbed immunologic mechanism, has been thoroughly reviewed in this country[3] though deep interest has been shown in France[4] and in England[5]. The process here would elaborate itself much as occurs in erythroblastotic disease. A variant of this disturbance would be drug sensitivity induced in the newborn through maternal administration. Here, the mechanism might involve actual suppression of platelet formation or a toxic effect on the newly formed platelet. It would appear essential that agglutination studies of platelets should be carried out in any given case of neonatal thrombocytopenia.

A third mechanism for the development of thrombocytopenia in the neonatal period involves associated blood changes originally described by Fanconi[6]. This defect has been associated within recent years[7,8] and the thrombocytopenia is actually part of a general bone marrow defect which includes myeloid and erythroid elements as well. This type of thrombocytopenia would be classified as megakaryocytic. The association of other congenital anomalies (particularly skeletal) have been noted and include cleft palate, renal and cardiac aberrations.

We have recently studied a newborn infant which showed rather striking features justifying a diagnosis of amegakaryocytic

* From the Newborn Service and the Dept. of Pathology, The Community Hospital, Indianapolis, Indiana.
** Hospital Pathologist.

thrombocytopenia. The early, minimal and rather transitory nature of the skin disorder which presented itself could easily have been ignored and spontaneous resolution have taken place. Failure to detect any hematological abnormality in the mother has added further interest to this case report.

CASE RECORD

Baby C. was born on January 5, 1959 and weighed 5 lbs. 4 oz. He appeared normal at birth and manifested no disturbance. A scattered, mild purpura developed over the body after 24 hours. This was noted particularly over the lower extremities and diaper region. The infant became irritable, showed some muscle stiffness and resented movement of the joints. Examination was essentially negative otherwise, and within 36 hours the purpura had disappeared.

The hemoglobin was 19.2 grams per hundred c.c. of blood, the WBC 8,550 per cmm with 77% polys, 6% bands, and 17% lymphocytes. The bleeding time was 2½ minutes, the clotting time 5 min. (capillary) and the platelet count 36,000 per cmm. The bone marrow on January 19, 1959 was normal with exception of total absence of megakaryocytes. (Figure 1)

Laboratory workup on the mother: Hemoglobin 12 grams per hundred c.c. of blood, WBC 20,275 per cmm. with 88% polys and 12% lymphocytes. The platelet count was 374,000. The urine was negative.

Course: The infant was treated with Achromycin gtts. IV and Neocortef, 5 mg. every 6 hours. Platelet counts were recorded as follows:

1- 6-59	36,000	1-15-59	101,000
1- 7-59	20,000	1-16-59	95,000
1- 8-59	36,000	1-17-59	88,000
1- 9-59	64,000	1-19-59	150,000 *Rx stopped*
1-10-59	52,000	1-20-59	104,000
1-11-59	46,000	1-21-59	101,000 (Hb.-14.5 Gm.)
1-12-59	89,000	1-23-59	139,000
1-13-59	103,000	(Discharged from hospital)	
1-14-59	107,000	3- 5-59	286,000

On March 10, 1959, the bone marrow was entirely normal.

The feeding consisted of half-skimmed milk, water and dextrimaltose. No disturbance was noted until January 13, 1959 at which

time respiratory distress and skin pallor led to the impression that aspiration of formula had occurred. Two days later, diarrhea occurred necessitating change of formula to protein milk. The progress was good thereafter with gain in weight to 6 lbs. 3 oz. at the age of 4 weeks. Splenomegaly was not demonstrated at any time.

TABLE I — BLOOD TYPING

		Father (O)	Mother (A)	Infant (O)
Rh^0	(D)	+	+	+
Rh^1	(C)	+	+	—
Rh^{11}	(E)	—	+	+
hr^1	(c)	+	+	+
hr^{11}	(e)	+	+	+
M		+	—	—
N		+	+	+
Kell		—	—	—
Duffy		—	—	—
CW		—	—	—

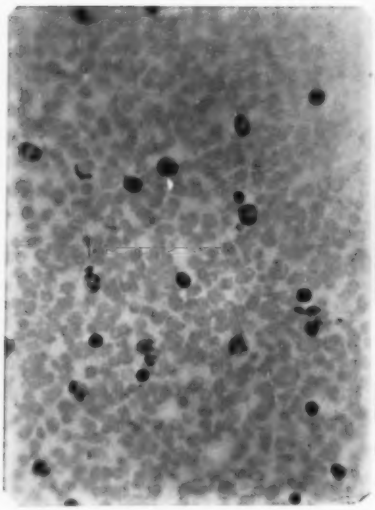

FIGURE I (A.)
Wright's Stain, mag. X275. (January 19, 1959). The bone marrow is normal except for the absence of megakaryocytes. An exhaustive search shows this to be a typical field.

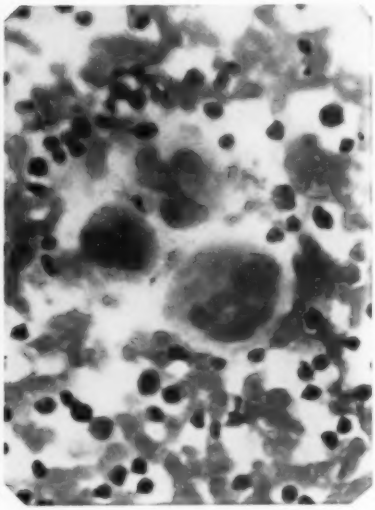

FIGURE I (B.)
Wright's Stain, mag. X300. (March 10, 1959). The bone marrow is now totally normal following return of the platelet count to satisfactory level. Note presence of three megakaryocytes in the center of the field.

TABLE II — PLATELET AGGLUTINATION STUDIES

COVERSLIP PREPARATION	Reading after incubation @ 37° C for 60 min.	Reading after incubation @ 0° C overnight	(Final Readings) Reading after re-incubation @ 37° C for 60 min.
Mother's platelets— Baby's platelets	absent	equivocal	present
Mother's plasma— Baby's platelets	absent	absent	absent
Mother's platelets— Baby's plasma	absent	absent	absent
Nembutal— Baby's platelets	absent	absent	absent
Nembutal— Mother's platelets	absent	absent	absent
Hykinone— Baby's platelets	absent	absent	absent
Hykinone— Mother's platelets	absent	absent	absent
Ergotrate— Baby's platelets	absent	absent	absent
Ergotrate— Mother's platelets	absent	absent	absent
Demeral— Baby's platelets	absent	absent	absent
Demeral— Mother's platelets	absent	absent	absent
Nembutal—Baby's plasma— Mother's platelets	absent	absent	absent
Hykinone—Baby's plasma— Mother's platelets	absent	absent	absent
Ergotrate—Baby's plasma— Mother's platelets	absent	absent	absent
Demeral—Baby's plasma— Mother's platelets	absent	absent	absent

DISCUSSION

The infant presented evidence, the reactions and criteria necessary for diagnosis of the rare form of thrombocytopenia—the amegakaryocytic type. The infant was born of a mother who at no time showed clinical or laboratory signs of thrombocytopenia. Blood type and blood platelet incompatibility were proven absent though the maternal-infant platelet preparation was read as equivocal.

We feel that little significance should be attached to the normal bleeding time since some infants blood freely only after un-

usual trauma. Akerrén and Reinand[9] reported normal bleeding time and prolonged clotting time in their cases. There was little bleeding in our patient though it is evident that some synovial reaction occurred as manifested by irritability and resistance to joint movement.

The specific depression in the bone marrow (absent megakaryocytes and failure of anemia to develop) removed the possibility of a Fanconi's hypoplastic anemia. Likewise, the lack of platelet incompatibility was demonstrated. It must be pointed out, however, that *this in no way eliminated the possibility of toxic effect of a drug upon megakarocyte activity* even though effect on erythroid and myeloid factors are absent. In fact, the return of the platelet count to a normal level within two weeks suggests that such a toxic effect may have been involved. The effect of the cortisone is difficult to evaluate and it is possible that spontaneous recession might have occurred with no therapy at all. It is difficult to withhold treatment in such a situation particularly where the conservative approach may have merit. Stefanini and Damesheck[10] state that "where megarycocytes are completely lacking, no effect on the platelet level" could be expected from splenectomy.

SUMMARY

A case of neonatal amegakaryocytic thrombocytopenia is reported which involved an infant of a normal mother. The disease was self-limited though cortisone was used for the initial 13 day period. No explanation could be found for the suppression of megakarocytes in the bone marrow though drug intoxication is suspected.

NOTE: *The authors are indebted to Miss Joan Hurst, R. N. and to Miss Annette Watts, M. T. for their assistance in gathering the clinical laboratory data.*

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The Community Hospital, 16th St. and Ritter Ave., Indianapolis, Indiana

ADVANTAGES OF A NEW SALICYLATE IN IMMUNIZATION REACTIONS IN INFANTS

EMANUEL DUBOW, M.D.*

New York

Reactions to immunizing injections used routinely in infants during the first six months of age, have been estimated to occur in from ten to twenty percent of cases. Even at the rate of only one in ten, the number of reactions, considering the relatively universal usage of these immunizations, runs in the millions. The reactions varying from mild to severe, are usually characterized by temperature elevations, crying, fretfulness and generally, evidences by the infant of discomfort and pain. Salicylates, especially aspirin, have found a wide-spread and effective usage in these reactions. However, the tablet type dosage form has been an inconvenience, especially in very young infants where accurate measurement of small dosages is desirable. Gastric irritation has resulted occasionally in young infants given aspirin, and vomiting is not infrequent following its usage. Also absorption rate from the stomach varies tremendously, but on the average does not give a therapeutic blood level in infants for at least thirty minutes and often considerably longer.

A newly synthesized preparation, choline salicylate†, appears to have properties which would make it particularly suitable for use in these reactions. It is liquid, making highly accurate dosage very simple, and it is very rapidly absorbed,[1,2] so that relief of symptoms should occur in a very short period of time. We endeavored to test the effectiveness of the drug and its tolerance by infants in the following clinical trials:

METHODS

Our test group varied in age from four to seven months. They were selected from a group of normal healthy infants who had had a reaction to their first injections of 0.5 c.c diphtheria-pertussis-tetanus vaccine. When the second injection was administered approximately four weeks later, the mothers were given a bottle of choline salicylate. With the onset of symptoms, they were in-

* Chief of Pediatrics, Beekman Downtown Hospital, New York.

† Made available by The Purdue Frederick Co. Medical Department in the form of Actasal [TM] Pediatric Drops

structed to administer fifteen drops of the medication and then to take rectal temperatures at ten-minute intervals, noting the time of a significant drop (arbitrarily selected by us as 0.5° F.). They were also asked to note whether there was a diminution in evidences of discomfort and malaise, and the lapse of time after administration at which this occurred. No attempt was made in this study to test the duration of the therapeutic effect.

Tweny infants were tested. The dosage of fifteen drops represented 0.127 Gm. of choline salicylate, which is equivalent to 1.48 grains of aspirin. The material was administered directly into the

. . . . . TABLE I . . . . .

Effect of Choline Salicylate on Immunization Reactions

Case	Age (Months)	Race Sex	SYMPTOMS AT ONSET Temp.	Other	TIME (minutes) for significant reduction temperature	SYMPTOMS 30 MINUTES AFTER THERAPY Temp.	Other	Result
1	4	W-F	101.2	Crying	10	100	0	Excellent
2	4	W-F	100.4	Fretful	*	100.8	Fretful	Poor
3	5	W-F	102.8	Flushed	10	99.2	0	Excellent
4	4 1/2	W-F	100.	Crying	20	99.6	0	Fair
5	6	W-M	101.4	0	30	99	0	Excellent
6	5	W-M	103.	Crying	10	101	0	Excellent
7	3 1/2	W-M	100.4	Fretful	10	99.4	0	Excellent
8	4	W-F	100.2	Fretful	10	99.6	0	Excellent
9	3	W-F	102.4	Crying	*	102	Crying	Poor
10	4	W-F	100.2	Fretful	10	99	0	Excellent
11	6 1/2	W-M	101.	0	30	100.2	0	Fair
12	5	W-F	100.4	Crying	20	99.4	Crying	Fair
13	5	W-M	100.8	Fretful	30	100	0	Fair
14	5	W-M	100.2	0	10	98.8	0	Excellent
15	3	W-F	103.2	0	10	99.4	0	Excellent
16	4 1/2	W-M	100.	Crying	10	99.4	0	Excellent
17	4	W-M	100.6	Fretful	*	100.4	Fretful	Poor
18	5 1/2	W-M	100.6	Crying	*	100.2	Crying	Poor
19	6	W-M	101	0	20	100.4	0	Fair
20	5	W-M	100.6	0	20	100	0	Fair

*No significant reduction in temperature during 30 minute period of observation.

mouth, undiluted. The period of observation for purposes of this study was thirty minutes. Only one dose of the medication was administered.

The results are shown in Table I.

RESULTS

The column in Table I headed "Result" represents the mother's subjective evaluations of the effect of the medication. Ten of the twenty considered the results excellent, six fair, and four poor. Of the poor results, cases two and nine probably vomited most of the medication an incidence of 10%.

TEMPERATURE

Sixteen of the twenty cases showed a drop in temperature of at least half a degree. Of the four who showed no temperature fall, two had vomited the medication (cases 2 and 9). One case, No. 15, showed a drop of 3.4° in ten minutes.

DISCOMFORT

Nine cases who demonstrated evidences of discomfort such as crying or fretfulness, were relieved by the dosage administered. Five cases were not relieved of these symptoms, but two of these had probably vomited the medication (cases 2 and 9). The other cases showed no symptoms of discomfort.

SPEED

Reduction of temperature or relief of discomfort or both occurred in nine cases within ten minutes, in four cases within twenty and three cases within thirty minutes.

CONCLUSIONS

Choline salicylate is an easily administered, well tolerated, and well accepted effective salicylate which lends itself well to therapy for febrile and other reactions following routine immunizations. (The rapidity of therapeutic effect noted in these trials is noteworthy.)

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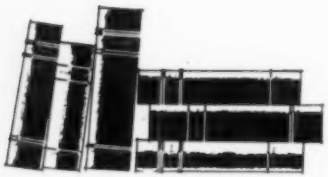
7 Lexington Ave., New York 10, N. Y.



. . . Books

Edited by

MICHAEL A. BRESCIA, M.D.

A Primer of Water, Electrolyte and Acid-Base Syndromes. By EMANUEL GOLDBERGER, M.D., F.A.C.P. Cloth. Pp. 322. Price $6.00. Lea & Febiger, Philadelphia 1959.

There are many books and pamphlets attempting to explain the intricacies of the blood electrolytes, pH and the acid-base balance and their interrelationships. However, to this reviewer Goldberger has presented the most logical approach to this admittedly difficult and intricate subject and has explained the physiological mechanisms involved so well that the subject almost becomes easy. For a clear understanding of this most important problem which would result in a more intelligent approach to the treatment of electrolytic imbalances, one should read this small volume carefully and keep it for ready reference. The book is devoid of unnecessary verbiage which enhances its practicality. M.A.B.

Environmental Influences on Prenatal Development. Edited by BEATRICE MINTZ. Cloth. Illustrated. Pp. 87. Price $3.00. The University of Chicago Press, Chicago, Ill. 1958.

This volume is another in a series of volumes containing the proceedings of the Developmental Biology Conference of 1956 which was held under the auspices of The National Academy of Sciences and the National Research Council. The papers and discussions contained in this volume were delivered at the Jackson Memorial Laboratory, August 2nd to 4th, 1956. The papers are particularly concerned with the factors both genetic and extragenetic that might effect the fetus as well as the multiplicity of factors that have teratogenic effects. These practically all deal with basic research in the lower species. But it is this essential research which will allow later extrapolation to the human problems of fetal environment. M.A.B.

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References: 1. Farah, L.: Internat. Rec. Med. 169: 379 (June) 1956. 2. Over 200 laboratory and clinical papers from 14 countries.
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oxytetracycline with glucosamine

CAPSULES

Continuation with oral Cosa-Terramycin every six hours will provide highly effective antibacterial serum and tissue levels for prompt infection control.

The unsurpassed record of clinical effectiveness and safety established for Terramycin is your guide to successful antibiotic therapy.

Supply:

Terramycin Intramuscular Solution*
100 mg./2 cc. ampules 250 mg./2 cc. ampules

Cosa-Terramycin Capsules
125 mg. and 250 mg.

Cosa-Terramycin is also available as:

Cosa-Terramycin Oral Suspension — peach flavored.
125 mg./5 cc., 2 oz. bottle

Cosa-Terramycin Pediatric Drops — peach flavored.
5 mg./drop (100 mg./cc.). 10 cc. bottle with plastic calibrated dropper

Complete information on Terramycin Intramuscular Solution and Cosa-Terramycin oral forms is available through your Pfizer Representative or the Medical Department, Pfizer Laboratories.

Contains 2% Xylocaine® (lidocaine), trademark of Astra Pharmaceutical Products, Inc.

Pfizer Science for the world's well-being™

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Brooklyn 6, N. Y.